PULSE OXIMETRY AND SIMULTANEOUS ELECTROCARDIOGRAPHY IN GERIATRIC PATIENTS UNDERGOING SURGERY

THESIS

For

DOCTOR OF MEDICINE

(ANAESTHESIOLOGY)

BUNDELKHAND UNIVERSITY
JHANSI (U. P.)

1993

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C E R T I F I C A T E

This is to certify that the work entitled "PULSE OXIMETRY AND SIMULTANEOUS ELECTROCARDIOGRAPHY IN GERIATRIC PATIENTS", which is being submitted as a thesis for M.D.(Anaesthesiology) by Dr. AJAY KUMAR SHARMA, has been carried out in the Department of Anaesthesiology, M.L.B. Medical College, Jhansi.

He has fulfilled the necessary stay in the department as required by the regulation of the Bundelkhand University, Jhansi.

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The techniques embodied in the thesis are undertaken by the candidate himself and the observations recorded have been periodically checked and verified by me.

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ACKNOWLEDGEMENTS

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Head of the Department cum Guide.

Dr. D.D. Verma, M.D.,D.A.,
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Mr. Om Prakash (Technician).

Mr. Zaheer Hasan (Clerk).

Mr. K.M. Thomas (Typist).

Mr. O.P. Yadav (Department boy).

I dedicate this work to my esteemed parents and teachers.

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I N T R O D U C T I O N

INTRODUCTION

In Geriatric patients there is an estimated 3-fold increase in mortality related to surgical procedures. Numerous data are available for review but essentially all interpretations reach the same conclusion. There are anatomic as well as physiological changes in cardiovascular and respiratory systems. The older patients have markedly reduced ability to respond to induced hypoxia or hypercapnia. Cardiac reserve is less. Hypertension is common and there is decreased ability to withstand stress, shock anaesthesia and surgery as compared to the younger person.

Severe arterial hypoxaemia may occur even during the most meticulously administered anaesthetics. Prolonged moderately severe hypoxia may be associated with pre-existing cardiac disease which is common in geriatric patients and a continuous, non-invasive monitoring of oxygen saturation by Pulse Oximetry may be of immense help in detecting hypoxaemia early.

Pulse Oximetry :

Pulse oximetry is a continuous and non-invasive monitoring of saturation of Haemoglobin in arterial blood

and recording of pulse. Pulse oximeter represents probably the most important advance in monitoring during anaesthesia since the introduction of the sphygmomanometer. The oximeters have all the advantages of a tissue plethysmograph and also display continuously the saturation of haemoglobin in arterial blood. In most devices there is a time-averaged digital or analogue display and an audible beat-by-beat sound, the tone of which is modulated by degree of haemoglobin saturation. These advices are accurate to one percent in the clinically important range of saturation (greater than 80%) (Yelderman & New, 1983; Taylor and Whitwam, 1986) have suitable response times, and are generally also superior plethysmographs in pulse detection (Griffithis et al, 1987). Although they detect haemoglobin saturation rather than O_2 tension, they have, for practical purposes, replaced percutaneous polarographic oxygen monitoring in adults.

The basis of oximetry is to shine light of particular wave-length through tissue and to measure the amount of light which is either absorbed or transmitted. The lobe or pinna of ear, the nail bed of the finger, or the wrist or ankle of a neonate, or even the septum of the nose may be used. The light is absorbed and scattered by the various tissues, skin, and haemoglobin in venous capillary and arterial blood.

The fundamental law governing the absorbance of light is known as Beer's law :

$$\frac{I_t}{I_o} = e^{Ecd}$$

where I_o = Intensity of the incident light

I_t = Intensity of the emerging light

e = Base of natural logarithms

c = Concentration of the substance through which the light passes in a path length of d.

Ecd = absorbance or optical density.

Therefore, for a given light path and for light of a particular wavelength the absorbance is a function of the (molar) concentration, c, of the substance in solution.

In the case of haemoglobin, the concentration may be determined by measurements made at two different wavelengths, at 805 nm (which is an isobestic point, i.e. at which the absorbance is the same for reduced haemoglobin as it is for oxyhaemoglobin) and at 650 nm where the difference in absorbance between the two forms is largest. This comparison permits the amount of oxyhaemoglobin to be estimated.

The pulse oximeter has been developed from the technological advances of microprocessors and light

emitting diodes. However, Beer's law does not strictly apply to non-invasive oximeters, and because saturation cannot be calculated theoretically it is estimated by an empirically derived alogrithm based on clinical data.

The pulse oximeter utilizes two light emitting diodes (LED_s) at the required wavelengths and analyses the changes in the light signal produced by the arterial pulsations by a rapid arithmetical subtraction process involving about 30 instantaneous calculations of saturation per second with averaging of 90 or 180 of these samples to produce a mean for 3 sec. or 6 sec. response.

Because pulse oximeters use two wavelengths only, other forms of haemoglobin e.g. methaemoglobin, carboryhaemoglobin and sulphaemoglobin, cannot be distinguished. Saturations may be under-estimated in patients with hyperbilirubinaemia (as bilirubin absorbs light at the relevant wavelength) and in patients with tricuspid incompetence (as the venules may also pulsate).

Electrocardiography :

The electrical activity of heart is recorded as electrocardiograph. The electrocardiogram may be used as a heart rate meter, to detect and characterize arrhythmias, and to provide some indication of myocardial ischaemia. Also, should a major problem occur unexpectedly,

the immediate availability of an ECG trace may be of great value. However, major disadvantages are that the ECG provides no indication of the adequacy of the circulation, and may provide a false sense of security if used as the only continuous monitor of the circulation.

Although ECG monitoring does provide additional information not provided by peripheral pulse-activated devices, it does not release the anaesthetist from monitoring the circulation continuously. Routine ECG monitoring is considered a 'minimum standard' by the Harvard group (in addition to some other monitor of the circulation) (Elchchorn et al, 1986), and has also been recommended in the U.K. An ECG should be used, only therefore, when other measures have been taken already to monitor a pulse continuously.

The goal of monitoring during anaesthesia is to detect untoward events and prevent them. An effective monitor assesses (ideally continuously) one or more markers of potential injury. The monitored information should enable the anaesthesiologist to alter therapy. Finally, the use of the monitor to manage therapy should objectively improve outcome. Keeping this in mind, it was therefore thought worthwhile to evaluate changes in oxygen saturation and electrocardiogram simultaneously in geriatric patients undergoing surgery under general anaesthesia and sub-arachnoid block.

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

Pulse oximetry represents the most important advance in monitoring during anaesthesia since the introduction of sphygmomanometer.

Pulse oximetry has its origin in the work of Nicolai who in 1931 applied Beer law to the transmission of light through the hand to study the dynamics of tissue oxygenation. He demonstrated that occlusion of the circulation produced an exponential fall in oxyhaemoglobin and rise in deoxyhaemoglobin.

Kramer in 1935 reported the continuous recording of oxygen saturation of blood flouring through unopened vessels using a spectrometric method. At the same time Matthes constructed the first device to measure oxygen saturation in vivo by transilluminating the ear.

Interest in aviators' oxygenation during World War II stimulated further development. In 1942 Squire, and later Goldie developed oximeters that set their zero value on tissue that had been compressed to squeeze out the blood, marking the beginning of modern pulse oximeters.

Milliken, in 1942, developed a light weight, practical aviation ear oxygen meter which he called an oximeter. Based on this work, Wood and Gracie, at the Mayo Clinic in 1949, built an oximeter with improved optics and an inflatable balloon that could make the ear bloodless for a reference setting. This device was manufactured by the Waters Company, and was extensively used in research, including evaluation of hypoxia during anaesthesia. More recently, Shaw, reported by Merrick and Hayes, developed a self calibrating ear oximeter using eight wavelengths of light that was commercially produced in 1976 and has become the standard against which other oximeters are judged.

In 1974, Aoyagi, as reported by Nakajima et al developed an oximeter that used the variations in volume which occurs with pulsatile arterial flow to obtain a signal representing oxygen saturation. Because of sensitivity to motion and because of its large size, the device was not widely used. Yoshia et al, in 1980, incorporated plethysmography and oximetry in an instrument that needed no calibration or heat to determine oxygen saturation. Using plethysmography to identify a volume change resulting from the arterial impulse, the device compared the light absorbance in the absence of a pulse - its zero - and in the presence of the arterial pulsation to determine arterial oxygen saturation. With this

integration of plethysmography and oxymetry and subsequent application of solid-state electronics, the pulse oximeter determines values within 2% of those measured in vitro, bringing us close to accurate, practical non-invasive assessment of arterial oxygen saturation. Motion artifact and difficulties with low perfusion states continue to limit performance of currently available devices.

Regarding the utilization of pulse oximeter in operation theatre, it is only since 1985 that it has found widespread use. The clinical utility of the non-invasive oximeter in operating theatre was rediscovered by William New and Yelderman (Evaluation of pulse oximetry. Anaesthesiology, 59 : 349-352, 1982).

Various workers have demonstrated the usefulness of pulse oximetry in detecting preclinical hypoxaemia and hence avoiding the hypoxic fatality to the patient.

Cote' and his workers described the incidence, duration, and severity of arterial oxygen desaturation as detected by pulse oximetry in infants and children. Additionally, in a cleverly designed single-blinded prospective manner they determined the impact of pulse oximetry information on the anaesthesia care team function.

A recent study of adult patients by Cooper et al, focusses upon an analysis of unanticipated,

undesirable events that were possibly related to anaesthetic management and required intervention in the recovery. In this study, the random use of pulse oximetry did not significantly increase the number of reports in the recovery room of hypoxic or hypoxaemic events, although the authors note that pulse oximetry may have provided information significantly earlier to clinician, thus preventing on hypoxaemic event (Cooper, J.B., Cullen, D.J., Neneskel, R., Hoagelin, D.C., Gevirtz, C.C., Csete, M., Venable, C.).

Another recent study by Severinghans and Naifeh describes in detail the performance of six commonly available pulse oximeter under conditions of sudden and profound transient arterial desaturation in adult volunteers (Anaesthesiology. 67 : 551-558, 1987).

In another recent study carried out by J.T.Moller, P.F. Jensen, N.W. Johannessen and K. Eskersen from Denmark it has been again proved that pulse oximetry reduces hypoxaemia in O.T. and recovery room (B.J.A., 1992 ; 68 : 146-150).

Regarding the use of electrocardiography in anaesthesia, J.B. Heard, A.E.Strauss and E.B.Krumbhaar, are considered pioneers who were the first to use electrocardiography in patients under anaesthesia in 1918.

Michael Johnson of University of Belfast had presented a paper on the usefulness of electrocardiography in 1948.

Lunn & Mushin in 1982 and Sykes in 1987 recommended for E.C.G. as essential part of monitoring of the patient under anaesthesia. Later on, E.C.G. recording was considered a minimum standard (in addition to another monitor of the circulation) by Harvard group (Eichhorn et al, 1986).

The pulse oximeter is an excellent monitor for transport from the operating room to recovery because of its portability and ease of use. In a study of American Society of Anaesthesiologists, Class I and II patients being transported while breathing room air, Tyler and associates (1985) found that 35% of their patients exhibited SpO_2 values below 90% during transport. This hypoxemia correlated with obesity and a pre-operative history of asthma. In a related study, Graham and colleagues (1986) found that 18 patients transported while breathing room air desaturated to an average SpO_2 of 89%, whereas 19 similar patients transported with supplemental oxygen experienced no major desaturations. In these two studies, the pulse oximeter firmly established the value of supplemental oxygen during transport to the recovery room. Hensley and co-workers (1986) also documented the necessity of administering supplemental

oxygen while inserting pulmonary artery catheters in pre-medicated cardiac surgery patients. In 12 of 20 patients, the SpO_2 values fell below 90% during catheter insertion, and SpO_2 fell to a low of 74% in 1 patient. Oxygen administered by nasal cannula at a rate of 4 L/min increased all of these patients' saturation to 95% or greater. Although these patients had various degrees of pulmonary disease in addition to their cardiac disease, all had adequate saturation values on arrival in the operating room before catheter placement. These data imply that nasal cannula oxygen should be administered to all patients in this situation.

The pulse oximeter is also a useful monitor of respiration in patients who are not in the operating room or intensive care unit setting. Choi and associates (1986) used SpO_2 to monitor post-cesarean section patients who were being treated with either epidural or parenteral narcotics. Each patient was monitored for approximately 1,000 minutes. Both groups exhibited an average of 3 to 4 minutes of desaturations below 90%, with no significant difference between the groups. The pulse oximeter was found to be a valuable monitor for these patients.

The study of patients with sleep apnea is another application of non-invasive oximetry. Strohl and Altose

(1984) used the Hewlett-Packard ear oximeter to monitor patients with sleep apnea during awake breath-holding maneuvers and during sleep. They found that the rate of fall of saturation was a function of the initial saturation at the onset of apnea. It was independent of whether the patient was awake or asleep and whether the apnea was obstructive or non-obstructive.

New applications for the pulse oximeter are being discovered on a regular basis. A pulse oximeter placed on the great toe has been used as an aid in cannulating the femoral artery in obese patients (Introna, R.P.S. & Silverstein, P.I., 1986). The pulse oximeter is now accepted as the primary indicator for the monitor of home oxygen therapy in patients with severe obstructive lung disease (Fulmer, J.D., and Snider, G.L., 1984).

If abnormal hemoglobin species can affect the accuracy of the pulse oximeter, one must also be concerned about dyes that are often injected intravenously during operation. These include methylene blue, indigo carmine, indocyanine green, and fluorescein. Scheller and Unger (1986) conducted a volunteer study in humans similar to the animal study by Sidi and co-workers (1986) and found that intravenously administered methylene blue caused very large decreases in SpO_2. In 1 volunteer the pulse oximeter reading fell to 1% after injection. Consistent

with the animal study, indo-cyanine green caused similar but less pronounced decreases in SpO_2 while fluorescein and indigo carmine had little effect on SpO_2.

Other technical problems in estimating SaO_2 by pulse oximetry have been reported. Motion artifact and external light interference have been reported by Brooks, T.D. et al, 1984. Kim and colleagues (1986) have reported that the pulsations in light absorbance may be primarily venous rather than arterial in origin. This implies that SpO_2 may read falsely low in circumstances leading to venous congestion, such as a dependent extremity.

Yelderman and New (1983) suggested that circumstances that reduce finger pulsation amplitude, such as hypothermia, hypotension, or administration of vasoconstrictor drugs, will adversely affect pulse oximeter accuracy. In the intensive care unit studies referred to earlier, several limitations were noted in this respect. In the study by Mihm and Halperin (1985), no pulse oximeter signal was obtained in 4 of 18 patients. Curiously, none of these patients was hypotensive or hypothermic. One of the patients had a history of vascular disease, and 2 patients were receiving dopamine, 1 with the addition of epinephrine. At the same time in this study the pulse oximeter was able to present a value on all patients who were hypotensive, most of whom were also receiving

vasopressors. Although these patients would be assumed to have high systemic vascular resistance, this was not established since cardiac output was not measured.

In other study, cardiac output measurements were determined along with systemic vascular resistance, body temperature, and hemoglobin (Tremper, K.K. et al, 1985). In 57 of 383 data sets, low signal values were obtained. In these data sets, 42% had a cardiac index less than 2.5 L/min/m^2, 16% had body temperatures less than 35°C, 9% had hemoglobin values less than 8 gm/dl, and 35% had a systemic vascular resistance greater than 2600 dyn/sec/m^2/cm^5. Although all these physiological values were out of the normal range, good pulse oximeter readings were obtained in other patients with more abnormal values. This study also evaluated "clinically significant false negative information". Clinically significant false-negatives were defined as data points in which SpO_2 values were 2% greater than SaO_2 values while the SaO_2 values were 95% or less. This occurred approximately 12% of the time and was associated with either abnormally high or abnormally low systemic vascular resistance. With regard to hypothermia, of the 15 data sets collected with a patient temperature less than 35°C, the pulse oximeter read a low signal nine times and read an average of 7% less than the arterial value the remaining six times (false-positive information). Results from these two

intensive care unit studies indicate that situations that cause low signal are apt to be associated with high or low systemic vascular resistance or hypothermia. With regard to the data from the study of Tremper et al (1985), Barker and Tremper (1987) used an older model pulse oximeter (Biox III, Ohmeda, Boulder, CO), and newer pulse oximeters with newer software may not yield the same results. It can also be seen that the pulse oximeter did obtain values over wide-ranges of cardiac output, systemic resistance, blood pressure, and temperature. These results indicate that there is some degree of patient-to-patient variability in the pulse oximeter's ability to analyse the pulse signal and produce an accurate saturation value.

Several authors have estimated anesthetic and surgical risk in the elderly patients. The multifactorial cardiac risk index of Goldman and colleagues identified increased risk in patients greater than age 70 years. Djokovic and Hedley-Whyte reported that the mortality in American Society of Anesthesiologists physical status 2 (based on age) octogenarians was less than 1.0%, though this is still perhaps 100-fold greater than that of younger patients. The age-related surgical risk in apparently healthy, elderly persons is due in part to chronic physiological abnormalities.

Physiological changes secondary to aging :

Aging inevitably results in a decline in physiological reserve.

With regard to pulmonary function, the progressive loss of alveolar surface area, termed ductectasia decreases the contact area between air and blood. This process, which resembles emphysema, leads to increased mismatching of perfusion and ventilation leading to increases in both shunting and dead space.

As surface area declines, the alveolar lining exerts less surface tension, thereby decreasing pulmonary elastic recoil. As a person ages, closing capacity (the lung capacity below which small airways begin to close) progressively increases, exceeding functional residual capacity by age 45 years in the supine and by age 65 years in the upright position. The resulting tendency toward airway closure is a major cause of the decline in arterial oxygenation that occurs with age. Other pulmonary changes that occur as a result of aging include decreased ventilatory response to hypercapnia and hypoxemia, decreased chest wall compliance, decreased muscle strength, and reduced maximal expiratory flow rates.

The age-related decline in pulmonary reserve generally limits the elderly less than does the deterioration of maximal cardiovascular performance,

whether due to age, chronic disease, or physical deconditioning (Spurgeon et al, 1983). Although resting cardiac output is well maintained, the cardiac response to stress diminishes with aging. When stressed, elderly patients increase cardiac output by increasing left ventricular end-diastolic volume and stroke volume rather than by increasing heart rate (Rodeheffer, et al, 1984). Myocardial compliance also declines with age, therby decreasing the margin of error in intravascular volume management. Aging also prolongs the time required for myocardial contraction and relaxation, resulting in several adverse effects, including higher heart chamber filling pressures, increased risk of subendocardial ischemia, and poor tolerance of tachycardia. Myocardial performance also appears to deteriorate because of abnormalities in calcium homeostasis and because of myocardial hypertrophy secondary to age-related increases in myocardial work (Geer, R.T., 1986).

As cardiovascular performance declines, so does the response to endogenous or exogenous B-adrenergic simulation (Lakatta, E.G., 1980). In contrast, the response to a-adrenergic drugs may be enhanced.

Concurrent disease processes compound age-related cardiovascular deterioration. Atherosclerosis generates obstructive lesions in both coronary arteries and

peripheral vessels. Myocardial infarction, hypertension, and cardiac valvular disease may precipitate congestive heart failure. An increased incidence of arrhythmias, especially heart block, atrial fibrillation, and premature ventricular contractions, may be due to the deposition of amyloid or calcium in the conduction system or to the atherosclerotic loss of elements of the conduction system with age (Geer, R.T., 1986).

Because the decline in cardiac and pulmonary reserve may be difficult to assess, quantitation of physiological abnormalities may require invasive monitoring Del Guercio and Cohn (1980) prospectively placed pulmonary artery catheters in 148 apparently fit patients greater than age 65 years. Of the patients studied, only 13.5% were physiologically normal; 63.5% demonstrated mild to moderate deficits, while 23% showed severe deficits. Of 34 patients with severe physiological compromise, operation was cancelled in 19, changed to palliative surgery under local anaesthesia in 7 others, and continued as planned in the remaining 8 patients, all 8 of whom died. This study suggests that many elderly patients have unrecognized diminished physiological reserve that contributes to peri-operative morbidity.

The autonomic nervous system and geriatric anesthesia :

With advancing age come declining physiological capabilities. The elderly patient's ability to preserve hemodynamic, respiratory, metabclic, and thermal homeostasis against perioperative assault is diminished. The mechanisms that maintain tissue perfusion and cellular function within acceptable limits depend, in a variety of ways, on the autonomic nervous system (ANS) and its effectors. Impairment of autonomic responses with age can result from deterioration of the sensor organs, afferent or efferent innervation, neurotransmitter production, autonomic receptors, cellular events distal to the receptor, or mechanical factors such as the vascular tree. Much research activity has been aimed at determining which functional changes predictably occur with age, independently of concurrent disease, and elucidating the causes of these changes at subcellular levels. Although the clinical implications of some research results remain speculative, the growing body of information on age-related changes in the ANS should help the anesthesiologist to better understand how the management of elderly patients and their responses to per-operative events will differ from that of young adults.

The most basic monitored parameters in anaesthesia, heart rate and blood pressure, are the foci of most

clinical studies of ANS changes with aging. Among healthy adults, resting heart rate and average heart rate do not seem to change with age. Resting heart rate is not identical to the intrinsic discharge rate of the sinoatrial node; it is modulated predominantly by tonic parasympathetic input. When parasympathetic and sympathetic blockade are imposed pharmacologically with atropine and propranolol, the intrinsic heart rate does decline with age (Kostis et al, 1982). Clinically important deterioration of cardiac pacing and conducting tissues occurs in some of the elderly, independently of ANS changes.

An age-related alteration in non-stressed heart rate that could involve the ANS is the loss of sinus arrhythmia. In young adults (without diabetes or other causes of autonomic neuropathy) the heart rate changes rhythmically with the phases of the respiratory cycle. Afferent inputs for sinus arrhythmia originate from pulmonary and intrathoracic vascular stretch receptors and the arterial baro-reflex receptors. As atropine has been found to ablate sinus arrhythmia, the efferent mechanism is considered to be the vagal slowing of the heart rate during exhalation. The variation of heart rate with respiration is consistently found to decline with age. In young adult diabetics with autonomic neuropathy (in which parasympathetic dysfunction seems to precede sympathetic), sinus arrhythmia is also lost.

In addition, the elderly manifest smaller heart rate increases after treatment with atropine than do young adults. For all of these reasons, some interpret the age-related loss of sinus arrhythmia to indicate a loss of parasympathetic influence on heart rate. If so, one also might expect the elderly to display diminished vagolytic tachycardia in response to pancuronium. Alternative explanations for the progressive loss of beat-to-beat heart rate variation could involve the aforementioned stretch receptors of baroreceptor reflex mechanisms (Vargas, E. & Lye, M., 1980; Davies, H.E.F., 1975; O'Brien, I. et al, 1986).

Among associated disorders, cardio-vascular diseases are particularly important; with the increased life expectancy, the number of patients suffering from cardiovascular diseases has steadily risen. According to Power's figures, the incidence of cardiovascular diseases is 6 percent in the seventh decade, and 100 percent in the eighth decade. Surgical mortality is considerably influenced by the presence of cardiovascular diseases. Nachlas et al (1961) observed a mortality of 6.6 percent in patients with cardiovascular diseases as opposed to 2.4 percent in patients without heart disease.

There is a high incidence of M.I. throughout the first post-operative week, and as most of the studies

in this field are retrospective, the exact moment of infarction often cannot be defined. This is especially true as a large number of the infarcts, varying from 21% (Tarham et al, 1972) to more than 60% are silent. The first indication of an infarction has often been cardiovascular collapse, or hypotension or arrhythmia which have led the attending physician to investigate. It is, therefore, perhaps not surprising that PMI have been revealed throughout the peri- and post-operative periods. In 1974-75 Steen, Tinker and Tarhan reported that 25% and in 1972 Plumlee and Boettner that 41% of M.I. were discovered during the operation, while both Tarhan and colleagues and in 1975-76, 1977-82 Rao, Jacobs and El.Etr reported a peak incidence on the 3rd day after operation.

Incidence of Peri-operative Myocardial Infarction :

Some studies do not distinguish between patients with and those without previous M.I., and report PMI rates varying from 0.08% to 24%, the highest tending to be in studies reporting mainly on patients with previous MI. In addition, the study by Baur, Nakhjavan and Kajani in 1965 reported an extreme 16% incidence in 150 patients selected at random from a surgical list. From these and other studies, it soon became apparent that whether or not the patient had had a previous MI was the factor most profoundly

affecting PMI rate. While Goldman and colleagues undertook a thorough multifactorial analysis of risk factors, including previous MI, from the outset most other authors have classified the patients into those with and those without previous MI. In this review, evaluation of other risk factors therefore refers mostly to patients with previous MI, as the infarction rate in patients without previous MI is so low that it is difficult to obtain data that are statistically evaluable.

Congestive heart failure :

This has also been identified as a risk factor. In addition, Pasternak and colleagues found a higher infarction rate in patients with a low pre-operative ejection fraction (less than 35%).

Angina pectoris :

Angina is the main symptom of coronary artery disease (CAD). Patients suffering from angina pectoris run a higher risk of cardiac complications such as MI than the general population. It is therefore, somewhat surprising that, although some authors report that many of the patients suffering a PMI had pre-operative angina most studies, including those with a more thorough multifactorial analysis, found stable angina not to be a significant independent risk factor.

Coronary artery disease shown by angiography :

In 1978 Mahar and colleagues reported that three vessel disease was a significant risk factor for developing PMI.

ECG changes before and after operation (excluding signs of previous MI) :

Many patients with CAD present with a normal resting ECG before operation. In patients admitted for peripheral vascular surgery, Tomatis, Fierens and Verbrugge in 1972 found that, among those with a normal resting ECG, 30% had severe CAD, with 75-100% obstruction of a major coronary vessel, and 14% had a 50-75% obstruction. Of these patients, some show typical ST-depression as a sign of myocardial ischaemia during exercise testing. Thus Cutler and colleagues in 1979 found that 73 of 100 patients admitted for vascular surgery had no ischaemic signs on a resting ECG, but 14 (19%) of these had ischaemic signs during exercise testing. Of the 27 patients with signs of ischaemia on the resting ECG, 18 (67%) had additional ST-changes during exercise. In these patients 48 operative procedures were performed, and PMI occurred in six, all of whom exhibited ischaemic signs on the exercise ECG. Arous, Baum and Cutler in 1984 similarly found that PMI occurred in 25% of patients with positive pre-operative exercise test who underwent major peripheral vascular surgery.

Von Knorring in 1981 reported that patients with pre-operative ST-segment and T-wave changes, but no other indication of CAD, had a higher incidence of subendocardial infarction than patients with previous MI from history or ECG (12% v. 6%). Others have studied selected groups of patients with ischaemic heart disease and abnormal ECG before operation, and reported high incidence of a further deterioration in the ECG after operation, with a 4-9% infarction rate.

Most authors have suggested that a pre-operative ECG indicating pre-existing ischaemic heart disease is linked to peri-operative cardiac complications. On the other hand, Goldman and colleagues found no ischaemic signs on the ECG, but pre-operative arrhythmias (both ventricular and supraventricular) or recent MI, to be independent predictors of post-operative cardiac complications. Breslow and colleagues (1986, Anaesthesiology, 64 : 398-402) reported that, while 19% (71 of 394 consecutive patients) had ECG abnormalities 1 hour after operation, nearly all were T-wave changes (flattening or reversal), and the incidence was not greater in patients with known CAD than in those without. In 70 of these 71 patients, the authors discovered no episodes suggestive of myocardial ischaemia in the post-operative period. They suggested that T-wave abnormality in the immediate post-operative period was a frequent

event not linked to the development of cardiac complications and probably not a marker of myocardial ischaemia. Thus there is considerable difference of opinion as to the importance of T-wave abnormalities, particularly when they are transient. Driscoll and colleagues used deeply inverted T-waves as the diagnostic criterion for PMI in 75% of the cases. These changes were normalized in all patients followed-up a few months later (four of nine patients). Thus the issue remains controversial.

High arterial pressure, as a transient event is an everyday occurrence in normal healthy individuals. Sustained high arterial pressure may be a secondary effect of many disease states, or it may occur independently of any specific disease state. In the latter case, the condition has been known as essential hypertension ever since Frank (1911) coined the phrase, but current convention designates the condition as primary hypertension, to differentiate it from secondary hypertension - that is, hypertension secondary to some specific cause.

Pickering (1968) emphasized that arterial pressure has a continuous distribution in the population, and there is no bimodal distribution that delineates normotensive and hypertensive sub-groups. Thus the definition of hypertension is based on a purely arbitrary choice of a dividing line as to what is normal. There is

no agreement as to the precise limit of normality, particularly with regard to systolic arterial pressures. Most authorities would regard a sustained diastolic pressure of 120 mm. Hg as distinctly abnormal, and many would regard a diastolic pressure of 110 mm.Hg as the lower limit for effective antihypertensive therapy. For the purposes of this discussion, a diastolic arterial pressure above 110 mm.Hg will be regarded as hypertension. As a justification to this value, we may consider the evidence of Goldman and Caldera (1979) that patients with diastolic arterial pressures below 110 mm.Hg are at no increased risk during anaesthesia and surgery.

Pre-operative hypertension :

This is defined very differently : either as a given arterial pressure value on a pre-operative evaluation or as patients receiving anti-hypertensive medication. There is a positive correlation between hypertension and ischaemic heart disease, and Prys Roberts, Meloche and Foex reported that patients with un-treated hypertension had the largest peri-operative haemodynamic alterations which, again, has been reported to correlate with a high PMI rate. It would therefore not be surprising if chronic hypertension also correlated with a high PMI rate, as reported by Eerola and colleagues, von Knorring, Steen, Tinker and Tarhan and Vormittag and colleagues.

On the other hand, there was no significant correlation in the study by Rao, Jacobs and El-Etr or that of Riles, Kopelman and Imparato or in the multivariate analyses performed by Goldman and colleagues and by Cooperman and colleagues. Thus this issue also remains controversial. In the first group of studies the statistical evaluation might have been inadequate, with no sequential multivariate analysis, and the latter studies reported on smaller numbers of PMI with a greater possibility of incorporating a type-II error statistically.

<u>Diabetes mellitus</u> :

Patients with diabetes have a two- to three-fold increase in prevalence of atherosclerotic disease. Most authors have therefore included diabetes in their investigations of risk factors for PMI. Only Driscoll and colleagues in 1961 have suggested a possible connection (but without statistical analysis), and the statistical data seem to refute such a connection.

<u>Age</u> :

The patient population is growing older and, with age, there is an increasing prevalence of ischaemic heart disease and associated diseases. This makes independent evaluation of age as a PMI risk factor more difficult, although in 1977 Goldman and colleagues

reported that age exceeding 70 yr. was an independent risk factor for development of cardiac complications. The population in a great number of the studies referred to here were patients with previous MI, and a patient who has experienced MI before the age of 30 yr. must have a serious underlying disease. It is, therefore, perhaps not so surprising that approximately 50% of the authors (retrospective), reported advanced age to be a risk factor, while the other 50% have not found a significant correlation.

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MATERIAL AND METHODS

MATERIAL AND METHODS

The study was conducted in the General Surgery O.T., Orthopaedics O.T. and Gynaecology & Obstetrics Operation Theatre of M.L.B. Medical College, Jhansi, during 1991-92.

Selection of cases -

The patients of ASA Grade I & II, between the age group of 50 to 90 years were selected from General Surgery, Orthopaedics and Obstetrics & Gynaecology wards. Patient's name, age, sex, body weight were noted. All the patients undergoing surgery were examined thoroughly in pre-anaesthetic clinic and wards and advised accordingly.

Routine investigations like estimation of haemoglobin, blood sugar, blood urea and routine and microscopic examination of urine were done. Electro-cardiogram and chest X-ray were also done of all the patients. The protocol for this study was institutionally approved and written consent was obtained for each patient.

The pulse oximeter used was the Minolta PULS-OX-6-A light source generated by two LEDs, wavelengths at approximately 660 nm and 940 nm and a photodiode

(finger probe) was mounted on a finger. No heating or "arterialisation" technology were required.

Electrocardiography was done by recording on each standard lead I, II, III, augmented leads aV_R, aV_L, aV_F and chest leads V_1, V_2, V_3, V_4, V_5, V_6.

Blood pressure recording was done by sphygmomanometer.

Every case was examined thoroughly before induction of anaesthesia. Pulse rate, B.P., and arterial oxygen saturation by pulse oximeter were recorded and ECG was recorded of each case before induction of anaesthesia.

Premedication :

In the pre-operative room each patient had intraneurestine with 18G I.V. canula. All the patients were premedicated accordingly. Inj. Atropine 0.6 mg was given intramuscular 45 minutes before induction of anaesthesia.

Technique of Anaesthesia - Following techniques were used for anaesthesia.

1. General anaesthesia -

$O_2 + N_2O$ + inhalational agent (Ether).

2. Spinal anaesthesia -

Subarachnoidal analgesia.

General Anaesthasia :

In general anaesthesia, pre-oxygenation was done for 5 minutes and patients were induced with a sleep dose of 2.5% Thiopentone sodium followed by 1-2 mg kg^{-1} body weight suxamethonium. IPPV started and followed by endotracheal intubation. Anaesthesia was maintained as :

$O_2 + N_2O +$ Ether.

Subarachnoidal analgesia -

In this technique 2.5 ml to 3.0 ml of 0.5% bupivacaine (sensorecaine) was injected by puncturing the duramater in L_3-L_4 or L_4-L_5 space by 21G or 22G lumbar puncture needle in right lateral or left lateral position with all aseptic precautions. After the establishment of the block, surgery was allowed.

Measurement / Assessment :

The pre. intra and post-operative evaluation was done by the same person. During operation, pulse, blood pressure, arterial oxygen saturation (SaO_2) by the pulse oximeter and electrocardiography was recorded and

the subjective assessment of blood loss during operation recorded.

Post-operative follow-up :

The patients were shifted to post-operative recovery room and monitored. The pulse rate, blood pressure, arterial oxygen saturation were recorded and electrocardiography was done in the immediate post-operative period.

Analysis of data :

The results obtained were compared using the simple statistical methods. The paired 't' test was used to compare the differences between the pre-, intra- and post-operative values in both the groups A & B and P value was taken from the chart of probability.

Statistical calculation :

1. Mean $\bar{X} = \frac{X}{n}$ where X = number of frequencies,
n = number of patients.

2. Standard deviation (S.D.) = $\sqrt{\frac{(X - \bar{X})^2}{n}}$

where X = number of frequencies,
$\bar{X}$ = mean,
n = number of patients.

3. Degree of freedom (d.f.) = n-1

4. Standard error of mean = $\frac{S.D.}{\sqrt{n}}$

where S.D. = Standard deviation

n = number of patients.

5. Paired t-test

$$t = \frac{\bar{d}}{sd / \sqrt{n}}$$

where $\bar{d}$ = $\frac{d}{n}$ (d = difference between X & Y)

n = number of patients.

s.d. = standard deviation of d-series.

6. 'P' value = taken from the chart of probability.

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O B S E R V A T I O N S

OBSERVATIONS

The present study has been carried out on a series of 30 cases admitted in M.L.B. Medical College Hospital, Jhansi.

Patients were divided into two different groups according to anaesthetic technique.

Group A :- Consisted of 10 patients, were given General Anaesthesia (Ether).

Group B :- Consisted of 20 patients, and were given subarachnoidal analgesia.

Table - 1

Age distribution of patients in each group.

Age (years)	Group A		Group B	
	No.	%	No.	%
50 - 60	1	10.0	2	10.0
60 - 70	7	70.0	7	35.0
70 - 80	2	20.0	7	35.0
80 - 90	-	-	3	15.0
90 - 100	-	-	1	5.0
Total	10	100.0	20	100.0
Mean ±S.E.	69.3 ± 1.45		68.8 ±2.04	

Table 1 shows that majority of patients were between 60 - 70 in group A (70%) and between 60 - 70 and 70 - 80 in group B that is 35% each. In group A, there were 10% patients in age group of 50-60, 70% in 60-70 years age group and 20% in age group of 70-80. In group B, the age group of 50 - 60 was having 10% patients while there were 35% patients each in age group of 60-70 and 70-80 years respectively. There were 15% patients in age group of 80-90 years and 5% in 90-100 years age group.

Table - 2

Weight of different groups.

Weight (Kg.)	Group A		Group B	
	No.	%	No.	%
40 - 50	-	-	2	10.0
50 - 60	3	30.0	8	40.0
60 - 70	7	70.0	8	40.0
70 - 80	-	-	2	10.0
Total	10	100.0	20	100.0
Mean±S.E.	57.3±1.53		54.6±1.85	

Table - 3

Haemoglobin (Hb%) of patients in each group.

Hb (gm%)	Group A		Group B	
	No.	%	No.	%
9 - 10	2	20.0	5	25.0
10 - 11	5	50.0	6	30.0
11 - 12	1	10.0	1	5.0
12 - 13	2	20.0	7	35.0
13 - 14	-	-	-	-
14 - 15	-	-	1	5.0
15 - 16	-	-	-	-
Total	10	100.0	20	100.0
Mean $\pm$S.E.	10.5 gm% $\pm$0.36		11.14 gm% $\pm$0.46	

Table - 4

ASA Grade distribution in different groups.

ASA Grade	Group A		Group B	
	No.	%	No.	%
I	8	80.0	16	80.0
II	2	20.0	4	20.0
Total	10	100.0	20	100.0

Table 2 shows that in group A, 30% patients were in weight group of 50 - 60 kg and 70% patients were in weight group of 60 - 70 kg. In group B, 10% patients were in each weight group of 40 - 50 and 70 - 80 kg, 40% each in group 50 - 60 and 60 - 70 kg respectively. Majority of the patients were between the weight group of 60 - 70 kg.

Table 3 shows the Haemoglobin (gm%) level of patients in each group. In group A, there were 20% patients in 9 - 10 gm% range, 50% patients were in 10 - 11 gm% range, 10% patients were in 11 - 12 gm% range, while 20% in 12 - 13 gm% range. None of the patients was in the range of 13 - 14 gm%, 14 - 15 gm% or 15 - 16 gm%.

While in group B, 25% patients were in the range of 9 - 10 gm%, 30% patients were in 10 - 11 gm% range, 5% patients were in 11 - 12 gm% range, 35% patients were in 12 - 13 gm% range and 5% patients were in 14 - 15 gm% range.

Table 4 shows the grade-wise distribution of the patients which was recommended by the American Society of Anaesthesiologists (ASA).

Majority of the patients were in the grade I. In the ASA grade I, in group A - 80%, group B - 80% patients were present, while in ASA grade II, there were 20% patients each in group A and B.

Table - 5

Change in pulse rate.

Study group		Operative Periods I(Pre.)	II(Intra)	III(Post)
A	Mean ±S.E.	84.1 ±2.45	86.1 ±5.65	88.3 ±1.43
B	Mean ±S.E.	87.8 ±3.24	77.5* ±3.25	91.6 ±3.16

* P ∠0.05

In table 5, the operative periods were divided into pre-operative (I), intra-operative (II), and post-operative (III) in different groups.

Table 5 shows that the mean pulse rate was 84.1, 86.1, and 88.3 in pre-operative, intra-operative and post-operative periods respectively in group A, while in group B, the pulse rate was 87.8, 77.5 and 91.6 in pre-, intra- and post-operative periods respectively.

In group B, the P-value was statistically significant in intra-operative period.

Table - 6

Changes in mean blood pressure (mm of Hg.) in each group.

Study group		Operative Periods					
		I (Pre)		II (Intra)		III (Post)	
		Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
A	Mean	126.6	79.4	123.0	75.0	128.0	76.0
$P > 0.05$	±S.E.	±4.37	±2.80	±4.79	±3.62	±2.54	±2.41
B	Mean	138.3	72.4	107.7*	71.5	126.0	77.5
	±S.E.	±2.59	±3.18	±1.83	±1.56	±1.87	±1.87

* $P < 0.05$

Table 6 shows the mean blood pressure changes in pre-operative, intra-operative and post-operative periods in all the two groups.

In group A, there was no significant change in mean blood pressure in intra-operative and post-operative periods as compared with the pre-operative mean blood pressure.

In group B, there was significant fall in mean blood pressure in intra-operative period (107.7/71.5) as compared with the pre-operative period (138.3/72.4). In post-operative period, again there was significant change in mean blood pressure (126.0/77.5).

Table - 7

Arterial oxygen saturation ($SaO_2\%$) in different groups.

Study group		SaO_2 I	SaO_2 II	SaO_2 III
A	Mean ±S.E.	96.40 ±0.63	91.70 ±1.78	96.60 ±0.88
B	Mean ±S.E.	96.40 ±0.44	89.60* ±1.79	96.35 ±0.59

* $P < 0.05$

Table 7 shows the mean arterial oxygen saturation ($SaO_2\%$) in pre-operative, intra-operative and post-operative periods in all the two groups.

In group A, there was no significant change in mean arterial oxygen saturation in intra-operative and post-operative periods as compared with pre-operative period.

While in group B, there was significant fall in mean arterial oxygen saturation in intra-operative period (89.6) as compared with pre-operative period (96.4). In post-operative period, there was no significant change in mean arterial oxygen saturation (96.35) as compared with pre-operative period. The values were clinically as well as statistically significant ($P < 0.05$) in the group B.

Table - 8

Effect of pulse rate, B.P. over the arterial oxygen saturation (SaO_2%) in different groups during the intra- and post-operative periods. All the values are in the mean value.

Study group	Pulse (Rate/min.)	B.P. (mm.Hg.)	SaO_2 (%)	Period
A	84.1	126/79.4	96.4	I Pre-operative period
B	87.5	138.3/72.4	96.4	
A	86.1	123/75	91.7	II Intra-operative period
B	77.5*	107.7/71.5*	89.6*	
A	88.3	128/76	96.6	III Post-operative period
B	91.6	126/77.5*	96.35	

* $P < 0.05$

Table 8 shows the effect of the pulse rate, blood pressure over the arterial oxygen saturation in intra-operative as well as the post-operative periods in groups A and B.

In group A, there was no significant effect of pulse rate and mean blood pressure over the arterial oxygen saturation in intra-operative period. While in group B, there was significant fall in arterial oxygen saturation (89.6%) with significant fall in pulse rate (77.5/min.) and mean blood pressure (107.7/71.5) in intra-operative period.

In post-operative period, again there was no significant effect of pulse rate and B.P. over the arterial oxygen saturation. While in group B, increase in pulse rate (91.6 per minute) and mean arterial pressure (126/77.5) lead to increase in arterial oxygen saturation (96.3%).

Table - 9

Effect of Hb% over the SaO_2% during intra- and post-operative periods in group A and group B.

Study group	Hb (gm%)	SaO_2 I	SaO_2 II	SaO_2 III
	9 - 9.9	97	93	94
	10 - 10.9	96.2	89.4	97.6
	11 - 11.9	92	92	92
A	12 - 12.9	98.5	96	99
	13 - 13.9	-	-	-
	14 - 14.9	-	-	-
	15 - 15.9	-	-	-
	9 - 9.9	96.4	97	95.4
	10 - 10.9	96	93.5	97.5
	11 - 11.9	96	88	96
B	12 - 12.9	96.1	84.7	96.5
	13 - 13.9	-	-	-
	14 - 14.9	100	76	97
	15 - 15.9	-	-	-

Table 9 shows no significant change of Hb% over arterial oxygen saturation.

Table - 10

Arterial oxygen saturation (SaO_2%) during the immediate post-operative period in different groups.

Study groups	SaO_2%		
	>90	85-90	<85
A (n=10)	10	-	-
B (n=20)	19	1	-

Table 10 shows the arterial oxygen saturation of the patients during the immediate post-operative period in all the two groups.

In group A, all the patients were having SaO_2 more than 90% while in group B only one patient was having SaO_2 90%, while 19 patients were having SaO_2 more than 90%.

Table - 11

Sex distribution of patients.

Groups	Total patients	Male		Female	
		No.	%	No.	%
A	10	5	50.0	5	50.0
B	20	11	55.0	9	45.0
Total	30	16	53.28	14	47.72

Table 11 shows the sex distribution in each group. In group A the male patients were 50% and in group B, male patients were 55%. In total 30 patients, the male to female ratio was 53.28% to 47.72%.

Table - 12

Period	E.C.G. finding	Group A No.of patients	Group A %	Group B No.of patients	Group B %
I Pre-operative	ST-T changes	None	-	None	-
	Heart Block	None	-	None	-
	Arrythmia	None	-	None	-
II Intra-operative	ST-T changes	None	-	None	-
	Heart Block	1	10.0	2	10.0
	Arrythmia	1	10.0	4	20.0
III Post-operative	ST-T changes	None	-	None	-
	Heart Block	None	-	None	-
	Arrythmias	None	-	None	-

Table 12 shows the ECG findings of patients in both groups A & B in pre-operative, intra-operative and post-operative periods.

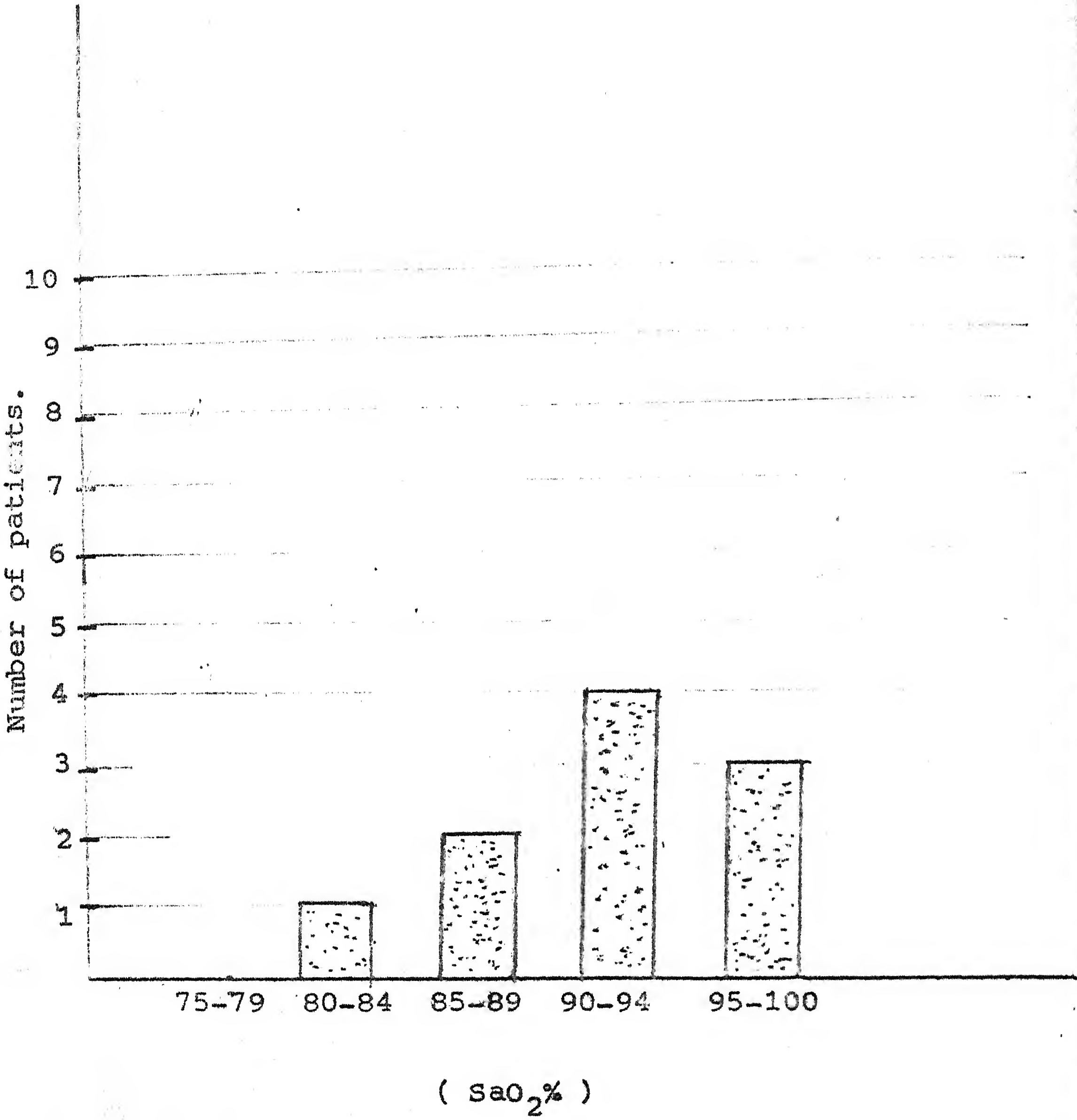

Fig. 1 : Lowest oxygen saturation under General Anaesthesia during intra-operative period.

ST-T changes were not found in both groups in pre-operative period. Heart block and arrythmias were also not seen in patients of both groups A & B in pre-operative period.

In intra-operative period, ST-T changes were not seen in both groups A & B. In group A, Ist degree of Heart block was observed in only one male patient, while in group B, Ist degree of heart block was recorded in 2 male patients. Premature beats were recorded in one female patient in group A while in 4 male patients in group B.

In post-operative period, ST-T changes, Heart block and arrythmias were also not seen in either group.

Fig. 1 shows the lowest oxygen saturation in number of patients under general anaesthesia (spontaneous ventilation) in group A. Only 10% of the total patient in group A had arterial oxygen saturation 80-84%, while 20% patients had 85-89%, 30% patients had 95-100% arterial oxygen saturation and 40% patients had 90-94% arterial oxygen saturation in intra-operative period.

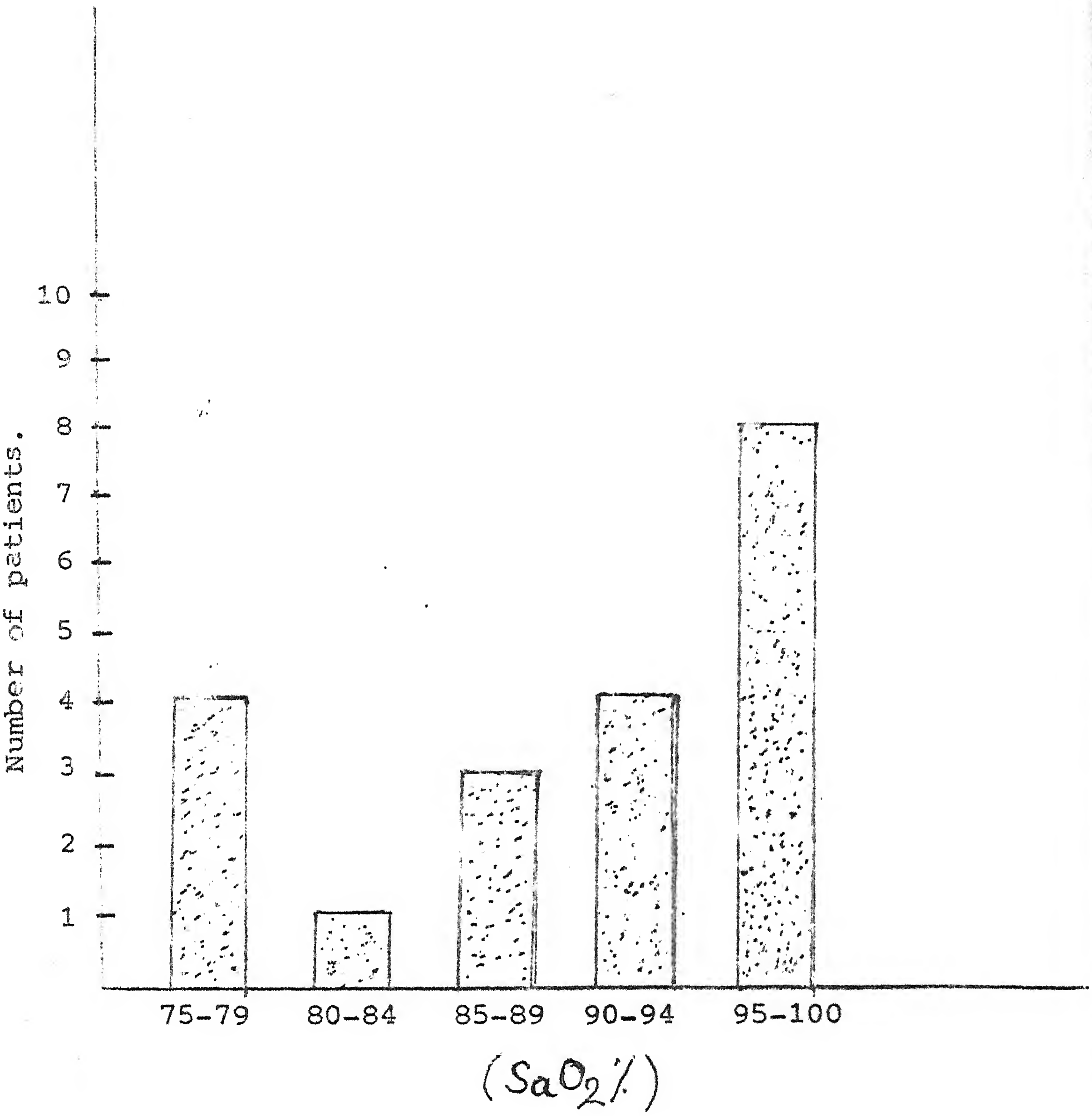

Fig. 2 : Lowest oxygen saturation under subarachnoidal analgesia.

Fig. 2 shows the lowest oxygen saturation in number of patients under subarachnoid analgesia in group B in intra-operative period. In this group, 20% patients had lowest oxygen saturation between 75-79%, 5% patients had 80-84%, 15% patients had 85-89%, 20% patients had 90-94% and 40% patients had 95-100% oxygen saturation.

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DISCUSSION

DISCUSSION

The older patient has a markedly reduced ability to respond to induced hypercapnoea or hypoxia, or both. There are physiological, pharmacodynamic and pharmacokinetic changes that accompany aging. It must be remembered that the elderly patient may have disease-related changes. The elderly are medically known for their large variability. Therefore, it is important that each patient be approached on an individual basis.

Aging per se most likely is not a major factor in predicting the risk of anaesthesia and surgery. Stephan (1984) reported that the mortality in 1000 patients 65 years of age or older is 5.8%. 85% of these patients had three or more pre-operative abnormalities. With this kind of data, one can see that physiological or disease state, or both is a better predictor of outcome than is age alone.

Therefore, in elderly patients it is important to monitor to detect untoward events and prevent harm.

The recent introduction of pulse oximeter has provided a continuous, non-invasive method to detect SaO_2. Pulse oximetry has proved an important monitor to detect arterial hypoxaemia as decrease in SpO_2 precedes changes in skin colour or haemodynamic variables. It has been again established that changes in heart rate and heart tones or ECG are late signs for the detection of arterial hypoxaemia (in only 17% of the major desaturation episodes did haemodynamic variables change) (Cote and co-workers, 1988).

The present study was conducted in the series of 30 elderly patients above 50 years of age. The monitoring done with the pulse oximeter (Minolta Puls-OX-7) and electrocardiography.

In this study, the SaO_2 was not affected by age of the patient as also confirmed by Nakatsuka and Bolling (1989).

The SaO_2 was significantly affected by the type of anaesthetic technique. Under general anaesthesia there was fall in SaO_2 (96.40% to 91.70%). But there was statistically significant fall in SaO_2 under subarachnoidal analgesia with bupivacaine (96.40% to 89.60%). These patients which were given subarachnoidal analgesia responded very well to the oxygen therapy. This finding

is not in accordance with the study of Nakatsuka and Bolling (1989) who did not find any significant changes in different anaesthetic techniques.

The SaO_2 was significantly affected by the fall in blood pressure during the intra-operative period and post-operative period. It was affected in group A under general anaesthesia non-significantly but in subarachnoidal block SaO_2 was significantly lowered (96.4% to 89.6%) with fall in blood pressure (138.3/72.4 to 107.7/71.5 mm of Hg.). It is so because immediately after giving the subarachnoidal analgesia there is sympathetic block which leads to fall in mean blood pressure. Due to reduction in the blood pressure there was tissue hypoxia or lowered arterial oxygen concentration towards the peripheral tissue. This episode was detected by finger probe and oximeter shown the fall in the SaO_2. With corrective measures the blood pressure was restored and SaO_2 again changed significantly in post-operative period (89.6 to 96.3%).

The SaO_2 was significantly affected by change in pulse rate in intra-operative and post-operative period. In general anaesthesia there was no significant effect of change in pulse rate over the SaO_2. In subarachnoidal analgesia there was significant change in SaO_2 (96.4 to 89.6) with change in pulse rate (87.6 per minute to

77.5 per minute) during intra-operative period. While in post-operative period there was again significant increase in SaO_2 (96.3%) with increase in pulse-rate (91.6 per minute from 77.5 per minute). Due to fall in blood pressure and pulse rate there was reduced cardiac output. Due to fall in cardiac output peripheral tissue perfusion was reduced leading to fall in SaO_2. On the contrary, the study carried out by Nakatsuka and Bolling (1989) shown that "the anaesthesia technique had no significant effect on SaO_2. Due to inhalation of 100% oxygen in post-operative period there was improvement in SaO_2 in both techniques of anaesthesia.

The ECG changes were not significant. The pre-operative ECG findings were normal in both the groups as the patients were selected from ASA grade I and II for elective surgery. Intra-operatively, due to fall in SaO_2, there were no changes whatsoever in ST-T segments in both the groups A and B as immediate remedies were instituted as soon as there was hypotension or fall in pulse rate. In group A only one patient out of 10 patients showed increase in P-R interval and in group B only 2 patients had increase in PR interval.

In group A ectopic beats were observed in one patient out of 10 patients under general anaesthesia with ether, while in group B under subarachnoidal analgesia

ectopic beats were observed in four patients out of 20. These supraventricular extrasystoles were disappeared themselves after few seconds of appearance. There was not another significant change in ECG recordings.

In post-operative period also there was no significant ECG changes whatsoever.

This study demonstrated that the SaO_2 was significantly affected by the fall in pulse rate and blood pressure in intra-operative period and post-operative period in patients in whom subarachnoidal block was given. In patients who were operated under general anaesthesia with ether, there was change in SaO_2 but not significantly. The study of Nakatsuka and Bolling (1989) confirmed that supplementation of O_2 inhalation decreased the incidence of hypoxaemia significantly.

Age, post-anaesthesia, body weight, sex, systemic diseases had no significant effect on hypoxaemia.

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CONCLUSION

CONCLUSION

After the study completed and data analysed, the following conclusion was derived that :-

1. The fall in pulse rate reduced the SaO_2% during the intra-operative period.

2. The hypotension reduced the SaO_2% during the intra-operative period.

3. The desaturation was more in subarachnoidal analgesia rather than general anaesthesia.

4. The finding of bradycardia (lowered heart rate) was similar in pulse oximeter and electrocardiogram.

5. The changes in rhythm were more common in patients under subarachnoidal analgesia.

6. The supplementation of oxygen inhalation during the intra-operative period increased the SaO_2 and greatly reduced the incidence of hypoxaemia in subarachnoidal analgesia.

With this study we conclude that pulse oximetry and electrocardiography must be essential for monitoring

during the subarachnoidal as well as general anaesthesia for elderly patients. Because decrease in SaO_2 detected by pulse oximeter precedes changes in skin colour and haemodynamic changes, so it is of immense help in reducing the incidence of hypoxaemia in elderly patients. And ECG changes are late signs for detection of arterial hypoxaemia so morbidity and mortality in elderly patients which have reduced adaptibility to hypoxaemia and hypercarbia.

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S U M M A R Y

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The pulse oximeter represents probably the most important advance in monitoring during anaesthesia since the introduction of the sphygmomanometer. It allows continuous registration of the arterial oxygen saturation by using the light absorption in a wave range between about 600 and 1000 nm. In addition, the peripheral pulse is determined by a plethysmographic method. It is a reliable monitor for oxygen saturation and provides trend information about circulation, both of which are particularly appropriate for patients breathing spontaneously.

The electrocardiogram is used as a heart-rate meter, to detect and characterize arrythmias, and to provide indication of myocardial ischaemia. Major disadvantages are that the ECG provides no indication of the adequacy of the circulation, and may provide a false sense of security if used as the only continuous monitor of the circulation.

Patients subjected to surgery and anaesthesia must rely on autonomic mechanisms to maintain homeostasis and adequate organ perfusion. In the elderly, many of these mechanisms are limited in the strength, the rapidity

and the range in which they can compensate for physiological stresses and trespass. Anesthesiologists rely on predictable results from manipulation of the ANS and its effector organs, but aging in itself seems to alter the responses to these pharmacological manipulations. The diseases and medications that often accompany old age are further confounding factors. Some of the changes found with age seem to arise from the ANS itself, while some more likely originate in structural alterations of the cardiovascular system. Although both basic and clinical research reports are rife with contradictions, a knowledge of ANS aging should help us to anticipate the particular responses and requirements of our older patients.

Concurrent disease processes compound age-related cardiovascular deterioration. Atherosclerosis generates obstructive lesions in both coronary arteries and peripheral vessels. Myocardial infarction, hypertension and cardiac valvular disease may precipitate congestive heart failure. An increased incidence of arrythmias, especially heart block, atrial fibrillation and premature ventricular contractions, may be due to the deposition of amyloid or calcium in the conduction system or to the atherosclerotic loss of elements of the conduction system with age. With cardiovascular deterioration, there is also age-related decline in pulmonary reserve.

The goal of monitoring during anaesthesia is to detect untoward events and prevent them. The monitored event should enable the anaesthesiologist to alter therapy. Finally, the use of the monitor to manage therapy should objectively improve outcome. Keeping this in mind, it was therefore thought worthwhile to evaluate changes in oxygen saturation and electrocardiogram simultaneously in geriatric patients undergoing surgery under general anaesthesia and subarachnoid block.

The study was conducted in patients of ASA grade I and II, between the age group of 50 to 90 years of age to be operated upon under general anaesthesia as well as subarachnoid block. Continuous SaO_2 monitoring was done with pulse-oximeter (Minolta Pulse-OX-7) and electrocardiography recording was done simultaneously.

After the study completed and data analysed, the following conclusion was derived that :-

1. The fall in pulse rate reduced the SaO_2% during the intra-operative period.

2. The hypotension reduced the SaO_2% during the intra-operative period.

3. The desaturation was more in subarachnoidal analgesia rather than general anaesthesia.

4. The finding of bradycardia (lowered heart rate) was similar in pulse oximeter and electrocardiogram.

5. The changes in rhythm were more common in patients under subarachnoidal analgesia.

6. The supplementation of oxygen inhalation during the intra-operative period increased the SaO_2 and greatly reduced the incidence of hypoxaemia in subarachnoidal analgesia.

With this study we conclude that pulse oximetry and electrocardiography must be essential for monitoring during the subarachnoidal as well as general anaesthesia for elderly patients. Because decrease in SaO_2 detected by pulse oximeter precedes changes in skin colour and haemodynamic changes, so it is of immense help in reducing the incidence of hypoxaemia in elderly patients. And ECG changes are late signs for detection of arterial hypoxaemia so morbidity and mortality in elderly patients which have reduced adaptibility to hypoxaemia and hypercarbia.

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